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Volume 42 | Issue 2

Article 1

1980

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Recommended Citation

Hahn, David L. and Glock, R. D. (1980) "Coccidial Infection in Neonatal Swine," *Iowa State University Veterinarian*: Vol. 42 : Iss. 2 , Article 1.
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol42/iss2/1

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Coccidial Infection in Neonatal Swine

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Summary

Coccidia have been implicated as another of the many pathogens responsible for scours in baby pigs. The clinical syndrome begins at about 5 days to 3 weeks of age and is similar to other enteritides of neonatal swine. The pigs begin to scour and do not grow well. In some cases, a mortality of up to 50% of those affected has been noted. Negative response to antibiotics normally employed in baby pig scours is often observed as another feature of the disease.

Effective diagnosis depends upon necropsy of acutely affected or freshly dead pigs. A presumptive diagnosis can be arrived at by making a smear of scrapings from an affected portion of gut. Definitive diagnosis of the disease should be based on histopathological examination of the affected gut for typical lesions and coccidial forms. For histopathology, the tissues should be preserved in 10% formalin and sent to a diagnostic laboratory.

Treatment, control, and prevention of the disease must be aimed at both the sow and the pigs. Sanitation of the farrowing environment is extremely important. Coccidiostats administered to the sow before, during, and after farrowing may decrease the load of organisms available to the pigs at birth. Coccidiostats administered to the pig may help decrease the morbidity and mortality of the disease in the neonate.

Introduction

Coccidiosis in swine has long been

recognized, but until recently it was thought to have little importance. The clinical syndrome described in veterinary texts consists of: diarrhea, weight loss, anorexia, and general unthriftiness. In veterinary texts, the disease is said to affect swine from 1 to 3 months of age. It is considered of much less importance in swine than in sheep and cattle because of its sporadic occurrence in the herd and the minimal death loss associated with the infection. Diagnosis of this syndrome in swine, described in veterinary texts, is based upon clinical signs in conjunction with identification of significant numbers of oocysts in the feces of the affected pig.

Recently, coccidial infection has been found in pigs much younger than those described above. In this syndrome, pigs from 5 days to 3 weeks of age are involved. Unlike the disease described in older pigs, this syndrome can produce a morbidity of up to 100% and a mortality as high as 50%. The clinical signs can be quite variable and, in younger pigs, resemble *Escherichia coli* infection or transmissible gastroenteritis.

Diagnosis by use of a fecal examination for oocysts is of little value since, in many cases, few oocysts are being produced during the active clinical phase of the disease.

The disease may be self-limiting in a herd, running its course in approximately 2 weeks, or it may continue to cause a problem lasting many months. Some seasonal variation has been noted in the disease, with most infections occurring from May to October. The infection has been observed both in confinement and in pasture farrowing operations.

Nine species of coccidia have been reported from swine in North America. Of these nine, *Isospora suis* and perhaps *Eimeria deblickei* seem to be most important in producing the clinical syndrome in neonatal swine. The pathogenesis of the disease

The authors thank Dr. G. W. Stevenson, Iowa Veterinary Diagnostic Laboratory, for assistance in preparation of this manuscript.

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produced has not been completely worked out. The organisms are assumed to enter the baby pigs' digestive tracts soon after birth through contact with sporulated oocysts present in the sows' feces. This conclusion was arrived at because of the synchronous nature of the developmental stages of the disease in some natural infections that have been studied.

Case Report

On September 17, 1979, a farm in north central Iowa was visited in order to evaluate a persistent scour problem in young pigs.

The pigs had begun scouring at 5 to 14 days of age. Some had been vomiting, and there had been a moderate death loss. The pigs had been given tylosin injectable antibiotic for 2 days, with little if any improvement being noted.

Several pigs from 3 different litters were selected for necropsy. Each litter was in a different stage of the clinical disease by the onset of diarrhea. Group A pigs were 8 days old and had just begun to scour. Group B pigs were 9 days old and had been scouring for 2-3 days and rarely vomiting. These pigs had been treated one day previously with tylosin. Group C pigs were 10 days old and had been treated with tylosin at 7 and 9 days of age.

The pigs were euthanatized and necropsied.

Group A

The intestinal contents were fluid, there was mild ascites, and some mesenteric lymph nodes were swollen. The jejunum and ileum had slightly thickened walls, with the lower jejunum and upper ileum containing a pale, fibrinonecrotic pseudomembrane (see Figure 1).

Group B

The pigs had watery intestinal contents. One of the pigs in this group had a thin, fibrinonecrotic pseudomembrane similar to that described in Group A.

Group C

The lower jejunum and upper ileum had mild mucosal edema with some mucoid exudate present in the lumen.

Results of Histopathological Examination of Tissues

Group A

The brain, liver, kidney, spleen, mesenteric lymph node, and heart had no microscopic lesions. The colon had some epithelial degeneration with moderate crypt dilatation. The villi in the jejunum and ileum were blunted. The epithelium of these villi contained coccidial forms which were mostly in the gamont (sexual) stages.

Group B

The brain, liver, kidney, spleen, mesenteric lymph node, and heart had no microscopic lesions. In the jejunum and ileum of these pigs, lesions varied from markedly shortened villi to frank necrosis of the mucosa, with an associated thick, fibrinonecrotic pseudomembrane. Moderate numbers of merozoites and some gamonts were present (Figure 2). The large intestine had mild mucosal edema and moderate crypt dilatation, with scattered epithelial cell degeneration.

Group C

The brain, liver, kidney, spleen, mesenteric lymph node, and heart had no microscopic lesions. The mucosa of the jejunum and ileum had marked degeneration of the villi, with many adherent, small, gram-negative rods.

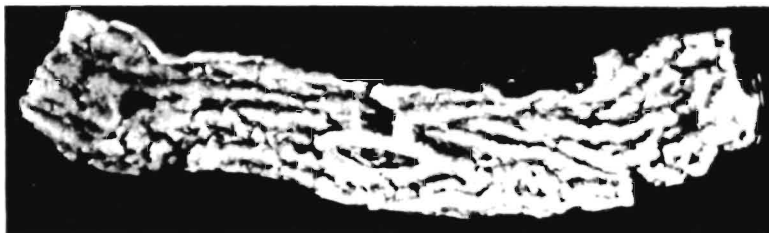


Figure 1: Gross lesions of swine coccidiosis, A fibrinonecrotic membrane covers the ileal mucosa.

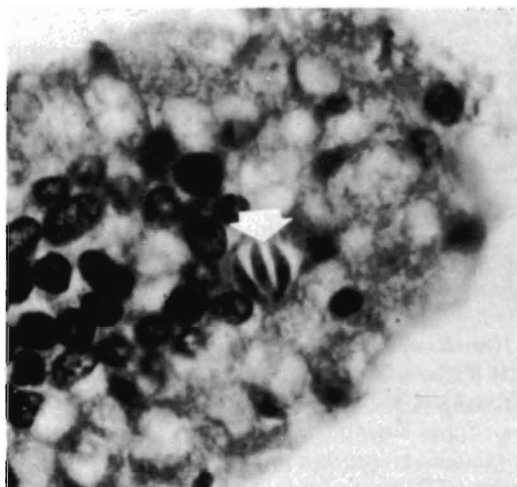


Figure 2: Villus infected with coccidia, presumed to be *Isospora suis*. Note the 3 plump merozoites within a vacuole (arrow). H&E, X650.

Additional Findings

Merozoites were observed in mucosal smears from the intestines of pigs in Group B after staining with Wright's stain (Figure 3).

The tissues and cecal contents were negative for TGE and rotavirus by fluorescent antibody technique and electron microscopy, respectively.

A high population of *Escherichia coli* was grown from the intestines of Group C pigs.

The diagnosis of this case was coccidiosis complicated by secondary *E. coli* infection in the Group C pigs.

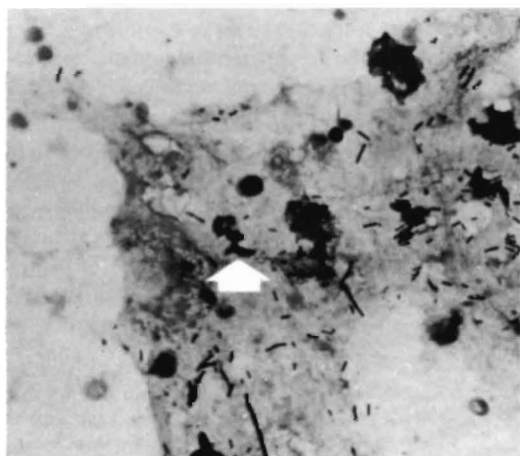


Figure 3: Mucosal smear showing 3 plump, crescentic merozoites. Bacilli can also be seen. Wright's, X30.

Discussion

Clinical signs of neonatal swine coccidiosis begin to develop 5 days to 3 weeks after birth. The signs can be quite variable, and differential diagnoses including TGE, rotavirus, *E. coli*, *Strongyloides ransomi*, and chronic *Clostridium perfringens* type C infection must be kept in mind. The primary feature of coccidiosis noted both from practitioners and from the literature is that affected pigs do not respond to antibiotic therapy. Pigs treated with tylosin, gentocin, tetracyclines, and other drugs commonly employed in baby pig enteritis had no response. Another clinical sign often observed is that the feces are fluid and grayish. Vomiting is another feature of the disease. Finally, pigs that recover often become very unthrifty. One practitioner put it this way, "Three-week-old pigs look like one-week-old pigs".

Upon postmortem examination, several features of the disease may be noted. First, there is usually a good milk curd in the stomach, and the lacteals are often empty. Second, there may be hyperemia of the small intestinal wall, especially in the ileum and jejunum. This can resemble that seen in milder forms of *Clostridium perfringens* type C infection. Third, in some cases a fibrinous to fibrinonecrotic exudate is adherent to the mucosa of the jejunum and ileum. Fourth, in milder or more chronic cases, catarrhal inflammation characterized by increased mucus in the lumen of the jejunum and ileum can be seen. Finally, in some cases no gross lesions can be identified upon postmortem examination except the presence of fluid-filled intestines.

Histopathological examination of multiple areas of the small intestine is extremely important for a definitive diagnosis of neonatal coccidiosis in swine. Thus, preservation of tissues for examination must be properly completed. If gross lesions in the gut are present, these portions and some less severely affected portions of gut should be harvested and put into formalin, making sure the mucosa is in contact with the formalin for proper fixation. If gross lesions are not present, several 1-inch sections from different levels of the gut should be fixed in formalin. In some pigs, it appears that rather short segments of intestine are affected, so careful examination and submission of numerous segments is important.

The lesions of coccidiosis are generally concentrated in the lower jejunum and upper ileum, with few if any lesions being noted in the duodenum or colon. In slight to moderately damaged areas, the villi are noted to be blunted, and there is an overlay of exfoliated epithelial cells. More severely affected areas will have complete loss of surface epithelium, with the denuded villi covered by a fibrinocellular exudate containing exfoliated cells. Merozoites measuring approximately 7.5 micrometers by 2.5 micrometers can be demonstrated in the damaged mucosa with hemotoxylin and eosin stains. The merozoites appear as crescentic or banana-shaped objects, each containing a nucleus that is darker than the cytoplasm of the structure. Male and female gamonts can also be demonstrated within the epithelium of some sections. The female gamonts are round, pale, and have a small, central, pink-staining nucleus. The male gamonts contain basophilic microgametes that resemble spermatozoa. In most cases, oocysts are not demonstrated in histological sections. It is important to note that the best diagnostic specimens are obtained from pigs very early in the course of the disease. Later, the damaged epithelium may slough, taking most of the coccidial organisms with it, thus making them difficult to find with histopathology.

Impression smears from the affected gut can be used in making a presumptive diagnosis of coccidiosis. Several levels of the jejunum and ileum should be selected for this procedure. The exudate overlaying the mucosa must be carefully wiped away. A glass slide is then touched to the mucosa. The impression smear is allowed to dry, and Giemsa's or Wright's stain is applied. The slide is then examined for the presence of basophilic, banana-shaped merozoites, gamonts, or oocysts.

Prevention of this disease must center around decreasing the amount of exposure to the pig. Since the baby pigs are apparently infected through exposure to organisms in sows' feces, an attempt should be made to keep farrowing crates free of feces during and after farrowing. Decreasing the number of oocysts present in the feces of the sow has also been advocated. Several coccidiostats have been used although none are specifically licensed for use in sows. Decoquate can be prescribed for administration to the sow for 2

weeks before and 1 week following farrowing at the rate of approximately 0.5 mg/kg per day (23 mg/100 lbs. of body wt) to help reduce exposure to the pigs. Amprolium has also been used at a rate of approximately 2 gm per sow per day.

Coccidiostats have also been used to prevent clinical signs in pigs that have been exposed to the organisms. The pigs may be given 1 ml of 9.6% amprolium solution orally for 4 to 5 days, starting at least 2 days before expected clinical signs. Favorable results have also been noted when 1 dose of the 9.5% amprolium was given 24-48 hours after birth.

This case report quite typically represents the syndrome now recognized as coccidiosis in neonatal swine. The presence of coccidial forms in the lesions, with absence of any other organisms able to produce lesions like those seen, lends support to the diagnosis of this syndrome. The differential diagnoses must be kept in mind when submitting tissues for diagnosis, since the signs of all these diseases can be similar. For this reason, tissues for diagnosis of TGE, *E. coli*, rotavirus, *Strongyloides ransomi*, and *Clostridium perfringens* type C infection should be included in the diagnostic submission. The importance of sanitation in prevention and control of this disease cannot be overemphasized.

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